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Human Exposure Assessment of Indoor Dust: Importance of Particle Size and Spatial Position

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Stapleton et al. (2012) reported that

> serum ΣpentaBDEs [sum of pentabrominated diphenyl ethers 47, 99, 100, and 153] were significantly correlated with both handwipe and house dust ΣpentaBDE levels, but were more strongly associated with handwipe levels ($r = 0.57$; $p < 0.001$ vs. $r = 0.35$; $p < 0.01$).

Here we propose an explanation for this phenomenon.

Toxicants are not distributed homogeneously in dust according to particle size; particle size distribution of settled dust varies with its spatial position. Thus, distribution of polybrominated diphenyl ethers (PBDEs) will vary with the particle size of dust and the spatial position of settled dust, as well as the location of PBDE sources, such that PBDE levels in settled dust on the floor will be different from those of settled dust above the floor (Björklund et al. 2012; Wu et al. 2010). Because of the spatial position of particles, humans are likely to be exposed only to particles of specific sizes. In addition, exposures to children and adults may be different because particles to which children and adults are exposed often have different spatial position and particle size distribution (Cao et al. 2012; Ruby and Lowney 2012). In addition, the reliability of human exposure assessments may be substantially influenced by between-room and within-room spatial variability of PBDE concentrations in indoor dust (Muenhor and Harrad 2012).

As reported in many other studies, Stapleton et al. (2012) reported that for dust sampling, they vacuumed "the equivalent of the entire floor-surface area for the room … by gently drawing the crevice tool across the top of all surfaces," and they selected fractions < 500 μm for their analysis. Only a few studies have demonstrated that particles > 250 μm are not appropriate for risk assessment of human exposures (Cao et al. 2012). Thus, if the dust samples from the house and from handwipes have different particle size distributions and are from different spatial positions in the indoor environment, it is inevitable that the PBDE levels in handwipes and house dust will be different and the correlation between PBDE in human serum and house dust will be weaker.

For human exposure assessment, we propose that indoor dust samples to be analyzed should be from relevant spatial positions and of specific particle size. By improving sampling strategies, we can obtain more accurate results and the correlations between PBDEs in human tissues and indoor dust will be much more accurate. In addition, settled dust should be sampled separately for adults and children.

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Human Exposure Assessment of Indoor Dust: Webster and Stapleton Respond

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We agree with Cao et al. that methods for sampling dust are insufficiently uniform between research groups and can be improved (Allen 2008a; Harrad et al. 2010). By using refined dust sampling methods we should be able to reduce exposure measurement error, likely random, leading to increased associations with exposure biomarkers. We have conducted several studies on polybrominated diphenyl ethers (PBDEs) investigating methods of dust sampling, the relationship between dust concentrations and potential sources of PBDEs, dust concentrations and biomarkers of exposure, and the use of handwipes as an intermediary step (Allen et al. 2008a, 2008b; Stapleton et al. 2008; Watkins et al. 2011, 2012; Wu et al. 2007). It is worth noting that dust sampling for environmental chemicals can have several purposes, including exposure assessment and characterization of sources. Dust sampling is also subject to a number of practical constraints such as sampling logistics and the requirement for sufficient mass of dust for adequate quantification of target compounds. We believe handwipes represent a more biologically relevant measure of indoor exposure for PBDEs than dust sampled from the floor of a room. Handwipes may also represent exposure experienced by direct contact with PBDE-treated sources. In addition, handwipes may integrate exposure across multiple microenvironments (Watkins et al. 2011, 2012). We agree that the dust particle size is likely to play a role in exposure to PBDEs, and this factor has received relatively little attention in the past. Recent work by Weschler and Nazaroff (2010) suggests that, on average, semivolatile organic compounds (including relatively more volatile pentaBDE congeners) are distributed in a room between air, dust, and surface films roughly as expected by equilibrium partitioning. The levels of pentaBDEs in all of these sampling media are therefore likely to show associations with body burden, although refinement of sampling methods may improve associations. The situation may be different for BDE-209, the main constituent of decaBDE that is essentially nonvolatile at room temperature. It may escape from products via friability rather than volatilization (Webster et al. 2009). The particle size distribution of BDE-209 in dust may be different than that of pentaBDEs. Finally, researchers and risk assessors estimate exposure to chemicals in dust by multiplying dust concentrations by highly uncertain exposure factors for dust ingestion (U.S. Environmental Protection Agency 2011). Additional research on dust ingestion factors is needed.

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Access to Chemical Data Used in Regulatory Decision Making

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It is clear from our commentary (Goldman and Silbergeld 2013), that we disagree with Lutter et al. (2013) about whether the public disclosure of all raw data used by the U.S. Environmental Protection Agency (EPA) for making regulatory decisions for chemicals is necessary to ensure the scientific basis for such decisions, and about the extent to which preemptive disclosure (prior to any request) is practical. However, the most important disagreement between us is the basis asserted by Lutter et al. in their commentary for this change in policy. Lutter et al. argued that it is necessary for the U.S. EPA—and anyone else who desires to do so—to reanalyze all data used in their assessments in order to "replicate" the findings and conclusions of the original investigators.

Lutter et al. (2013) repeatedly used the terms "replicability" and "replication" as synonymous with an "independent analysis" of raw data from an existing study. Replication in science is quite different; it involves performance of an independent study with the same hypothesis and then testing the extent to which this independent study reaches the same conclusions. Recalculation of study statistics or other reanalysis of an existing study data set is not a replication. Designing and conducting a replication study does not require access to raw data from the original study; this would abrogate the concept of independence. Moreover, an independent study will by definition utilize different sets of animal models or human populations, and as a consequence may employ different statistical techniques.

Their second argument is that disclosure of raw data will assist in identifying sources of scientific bias. We consider this unlikely because the most important sources of bias are usually related to problems in study design or limitations of the data collected. This is not identifiable through data recalculation; however, this type of bias can usually be identified in the text of the original study publication.

Lutter et al. (2013) noted (correctly) that applicants to the U.S. EPA for pesticide registrations must provide raw data from regulatory testing as part of the package submitted to the U.S. EPA. This is a very special case, in that these studies are neither peer reviewed nor accessible to the public because of the protection sought by industry and extended by law for confidential business information (CBI). The assumption of bias related to these studies is not unreasonable, given that they are conducted by or on behalf of commercial entities seeking to obtain pesticide registration. These studies are rarely published in the scientific literature or in any way subject to independent peer review other than review by the U.S. EPA. Many scientists and public policy practitioners consider the CBI cloak as a major impediment to transparency and confidence. Industry could demonstrate their commitment to transparency by declining this protection, thereby increasing the confidence of all.

Finally, Lutter et al. (2013) attempted to support their proposal by claiming that journals [*Nature* and the *Proceedings of the National Academy of Sciences of the United States* (*PNAS*)] and an expert body (the Bipartisan Policy Center) agree with them. However, these bodies have neither supported the concept of requiring that all raw data be reported to the the U.S. EPA nor that the U.S. EPA carry out its own independent recalculation. Rather, *Nature* and *PNAS* require authors to agree to make data sets (as well as materials and protocols) available to editors, and to others, upon request (Nature Publishing Group 2012; PNAS 2012). One of us (L.R.G.) was a member of the Science for Policy Project; its final report (Bipartisan Policy Center 2009) also recommended this practice. Many journals require data, such as DNA and protein sequences, macromolecular structures, microarray data, and crystallographic data, to be made available on publicly accessible databases, but most of these are not "raw data" in the sense that Lutter et al. proposed. *Nature* also recommends that authors submit clinical trials data to external clinical trials databases (Nature Publishing Group 2012).

In summary, we disagree with the argument that raw data from every study used by the U.S. EPA to support a regulatory assessment should be made available to the agency and to the public. This proposal does not serve the purpose of "replication" or identification of bias, as asserted by Lutter et al. (2013). In practice, it may generate obstacles to good science and discourage researchers from studying issues of importance in environmental health. This proposal would also limit the U.S. EPA from using the results of research published in the peer-reviewed scientific literature by placing studies off-limits if the authors did not submit raw data sets to the the U.S. EPA.

Finally, there is no obvious need for these changes. When the U.S. EPA has determined a need to reanalyze data, the current regulatory practice has not impeded such activities. Past history indicates that difficult cases are rare and do not warrant an intrusive and burdensome new requirement for the automatic submission of data from all studies.

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